BULLETIN OF
THE NEW YORK ACADEMY
OF MEDICINE

VOL. 44, NO. 5 MAY 1968

SIGNIFICANCE OF SPLIT HEART SOUNDS IN CHILDREN WITH WOLFF-PARKINSON-WHITE SYNDROME*

RAMON RODRIGUEZ-TORRES, ALICE C. YAO, AND JOSHUA LYNFIELD

Division of Cardiology, Department of Pediatrics
State University of New York Downstate Medical Center
Brooklyn, N. Y.

THE preexcitation syndrome was originally described by Wolff, Parkinson, and White.[1] Engle,[2] Schiebler and his co-workers[3] and, more recently, Swiderski and his co-workers[4] are notable among many who have since contributed to a better understanding and natural history of this syndrome in children.

Abnormal splitting of the heart sounds has been reported in normal children with Wolff-Parkinson-White syndrome (WPW).[5] The purpose of the present study is to determine whether splitting of the heart sounds is reliable for the diagnosis of associated heart disease in patients with WPW.

MATERIALS AND METHODS

The electrocardiograms of 4,400 patients who had been examined in the Pediatric Cardiology Department during the preceding five years (1962-1966) were reviewed. Six typical records that filled the electrocardiographic[6] and vectorcardiographic[7] criteria for type B WPW were found. One case was eliminated from this study because no phono-

*Presented at a meeting of the Section on Pediatrics, January 13, 1966.
This investigation was supported in part by Public Health Service Research Grant 12-8316.

cardiogram was available or obtainable. A routine 12-lead scalar electrocardiogram was recorded in each patient. Vectorcardiograms were obtained by the Frank system,[8] employing a Sanborn vector amplifier coupled to a Sanborn Viso-scope and photographed by a Polaroid camera.

Phonocardiograms were taken in four patients by means of a four-channel photographic recording system (Sanborn Model 564 Poly Beam). In one patient the first recording was obtained by a Sanborn Model 62 Twin Beam. Phonocardiograms were taken at 75, 100, and 200 mm./sec. They were recorded over the apex, the 5th left intercostal space near the sternum, the 3d left intercostal space near the sternum, and the 2d left and 2d right intercostal space near the sternum, at frequencies of 50, 100, 200, and 400 cycles per second. In Cases Nos. 4 and 5 two microphones were used simultaneously at the 5th left intercostal space and apex or at the 2d left intercostal space and 2d right intercostal space, in order to identify the valvular component of the heart sounds.

The phases of respiration were registered by a marker manipulated by an observer who watched the movements of the chest.

Lead II of the electrocardiogram was taken simultaneously with the phonocardiogram as reference. The standardization was increased (1 mv = 2 cms) in order to analyze the changes of the delta wave with respiration.

Indirect carotidograms were taken in all cases except No. 2; WPW was present at the time of recording. Measurements of the time interval were made in each phonocardiographic tracing from the beginning of the P wave to the mitral and tricuspid component of the first heart sound and to the aortic and pulmonic component of the second heart sound. When identification of each valvular component was not possible, the measurement was obtained from the beginning of the P wave to the main deflection of the first or second heart sound.

As demonstrated by March and his co-workers,[9] the P wave is preferable to the Q wave as a reference point in the presence of WPW, in view of the changes of the QRS complex in this syndrome.

The degree of splitting of the second heart sound was analyzed by measuring the time interval between the R wave of the electrocardiogram and the aortic and pulmonic component of the second heart sound. The values obtained by this method were compared with similar measurements made by Castle and Jones[10] in normal children.

TABLE I—SUMMARY OF CLINICAL FEATURES
FIVE CHILDREN WITH TYPE B WOLFF-PARKINSON-WHITE SYNDROME

Case No.	*Age*	*Sex*	*Clinical diagnosis*	*Heart sounds splitting*	*Arrhythmias*
1	30 months	M	Normal heart	Paradoxical	PAT†
2	6 months* 6 years**	F	Pulmonary stenosis	Single* Normal**	PAT†
3	10 years	M	Rheumatic aortic insufficiency	Normal	None
4	9 years	F	Rheumatic mitral insufficiency	Single	PAT†
5	18 months	F	Congenital mitral insufficiency	Single	None

*Wolff-Parkinson-White syndrome present.
**Wolff-Parkinson-White syndrome absent.
†Paroxysmal supraventricular tachycardia.

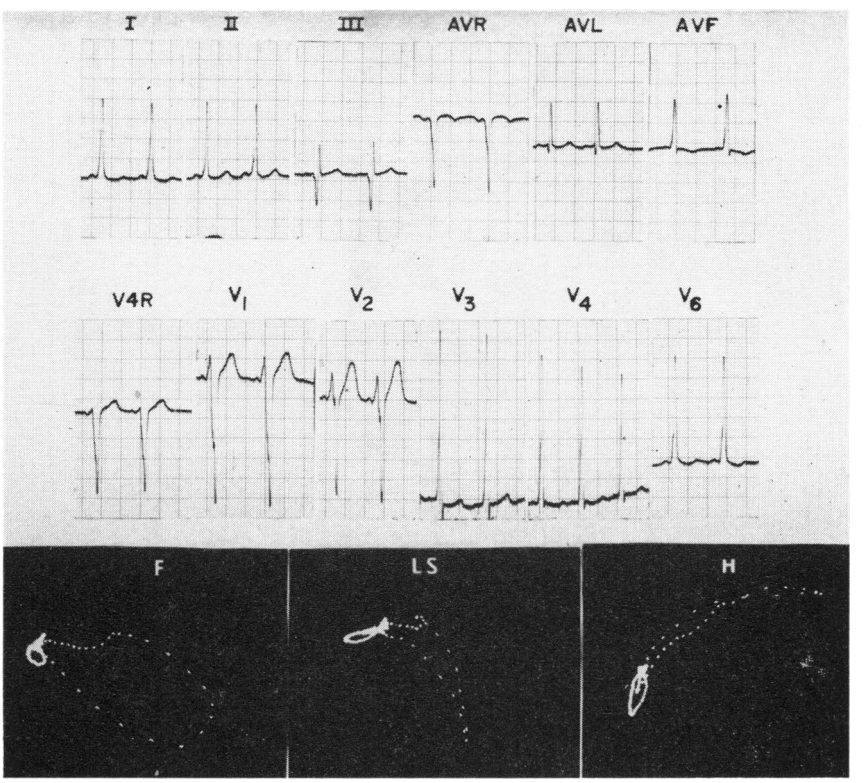

Fig. 1. Electrocardiogram and vectorcardiogram of Case No. 1 showing the findings characteristic of type B Wolff-Parkinson-White syndrome.

TABLE II—SUMMARY OF ELECTROCARDIOGRAPHIC AND VECTORCARDIOGRAPHIC FINDINGS

Case No.	*P wave AMP mm.*	*P wave duration seconds*	*A P degrees*	*P-R interval seconds*	*QRS duration seconds*	*Mean Δ vector frontal degrees*	*A QRS frontal degrees*	*Mean Δ vector horizontal degrees*	*A QRS horizontal degrees*
1	1.0	0.08	+60	0.08	0.12	—20	+30	—60	—45
2	1.5	0.08	+60	0.08	0.10	—5	—30	—15	+10
3	1.0	0.08	+60	0.08	0.08	0	+10	+30	+30
4	2.0	0.10	+60	0.08	0.12	+60	+60	—75	—60
5	1.0	0.08	+60	0.08	0.08	+60	+60	—70	—60

TABLE III—SUMMARY OF DIFFERENT MEASUREMENTS MADE IN THE PHONOCARDIOGRAM ON EACH PATIENT DURING INSPIRATION AND EXPIRATION

EKG-PKG Measurements in seconds	*Case No. 1*		*Case No. 2**		*Case No. 2***		*Case No. 3*		*Case No. 4*		*Case No. 5*		
	Insp.	*Exp.*	*Insp.*	*Exp.*	*Insp.*	*Exp.*	*Insp.*	*Exp.*	*Insp.*	*Exp.*	*Insp.*	*Exp.*	
P-S1	0.22	0.20	0.14	0.14	0.18	0.18	0.23	0.23	0.24	0.22	0.17	0.16	***
P-S2A	0.44	0.45	0.39	0.39	0.46	0.47	0.59	0.60	0.49	0.50	0.40	0.40	***
P-S2P	0.42	0.41	0.39	0.39	0.51	0.48	0.61	0.60	0.49	0.50	0.40	0.40	***
R-S2A	0.39	0.36	0.56	0.54	0.41	0.41	0.43	0.36	0.35	0.34	0.41	0.37	****
R-S2P	0.36	0.31	0.56	0.54	0.48	0.43	0.47	0.37	0.35	0.34	0.41	0.37	****
S1M-S2A	0.29	0.33	0.34	0.34	0.34	0.36	0.34	0.36	0.35	0.34	0.38	0.38	****
S1T-S2P	0.33	0.30	0.34	0.34	0.38	0.38	0.38	0.36	0.35	0.34	0.38	0.38	****
P-Δ	0.14	0.16	0.14	0.12	—	—	0.18	0.16	0.16	0.14	0.16	0.14	****
Δ Ht (mm.)	11	16	2.5	2	—	—	6	4.5	13	11	21	19	

*When WPW was present.
**When WPW was absent.
***Uncorrected for heart rate.
****Corrected for heart rate.

The duration of the mechanical systole of the right ventricle and left ventricle was determined by measuring the distance on the phonocardiogram from the main deflection of the first heart sound, when the mitral or tricuspid component could not be identified, to the aortic and pulmonic component of the second heart sound. The result was compared with the value obtained in normal subjects by Gray.[11]

The interval between the beginning of the P wave and the end of the delta wave, and the amplitude of the delta wave, were measured in each case. Each value (see Table III) is the mean result of at least three determinations taken on inspiration and three on expiration on each patient.

Results

Clinical Features

Table I summarizes the significant clinical findings in the five patients.

Electrocardiographic and Vectorcardiographic Findings

The electrocardiograms were similar in configuration in all patients. All belonged to type B WPW syndrome, according to classification of Massie and Walsh[6] (Figure 1). Table II gives relevant data obtained from the electrocardiograms and vectorcardiograms of the five patients.

Phonocardiographic Findings

First heart sound. Normal position of the mitral and tricuspid components of the first heart sound was found in one patient. The tricuspid component preceded the mitral component (paradoxical splitting) in one patient. No separation of the valvular components of the first heart sound was found in the other three patients.

Second heart sound. Physiological splitting of the second heart sound was found in Case No. 3. In Case No. 1 the pulmonic component preceded the aortic component (paradoxical splitting), as shown in Figure 2. No separation between the aortic and pulmonic component was present in Cases Nos. 4 and 5. In Case No. 2 no separation between the aortic and pulmonic component occurred when WPW was present, but it was possible to identify the two components when the preexcitation syndrome had disappeared (Figure 3).

Diastolic gallop. A protodiastolic and presystolic gallop oscillations were recorded in one patient (Case No. 4) during heart failure. Con-

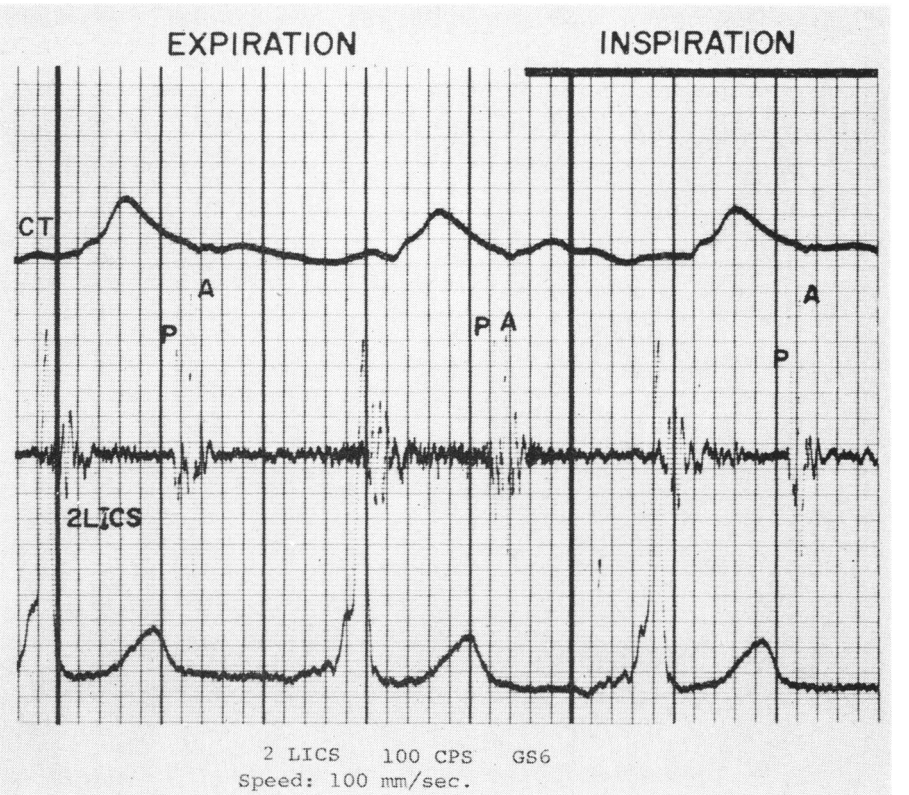

Fig. 2. Phonocardiogram of Case No. 1 recorded at 2d left intercostal space demonstrating wider separation between the aortic and pulmonic components of the second heart sound (paradoxical splitting of the second heart sound). Note also the increase in amplitude and duration of the delta wave on expiration compared with inspiration.

trary to what has been reported by Aravanis and his co-workers,[12] no abnormal oscillations were consistently recorded during the time of inscription of the delta wave.

Murmurs. Heart murmurs were heard and recorded in all five patients, including the one who had an anatomically normal heart. In this patient a short systolic murmur was recorded in the 3d and 4th left intercostal spaces. The patients with rheumatic and congenital mitral insufficiency had high frequency pansystolic murmurs at the apex. A decrescendo high-frequency early diastolic murmur was recorded over the 3d left intercostal space in the patient with rheumatic aortic insufficiency.

In Case No. 2 (pulmonary stenosis), a crescendo-decrescendo high-frequency systolic murmur was recorded over the 2d left intercostal

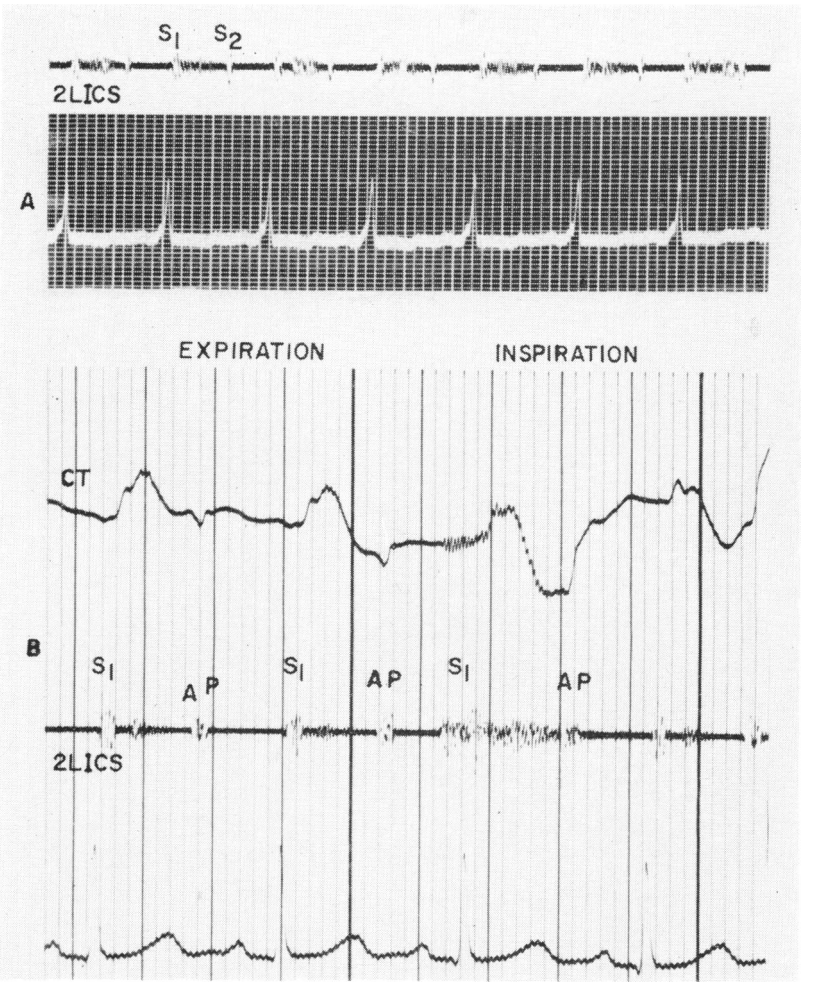

Fig. 3. *A*) Phonocardiogram of Case No. 2 at the time when Wolff-Parkinson-White syndrome was present, demonstrating the presence of a single second heart sound. *B*) Phonocardiogram obtained in the same patient five years later when Wolff-Parkinson-White syndrome was absent, demonstrating the presence of physiological splitting of the second heart sound.

space and left sternal border. This murmur was recorded with higher amplitude when WPW was present than when it was absent.[13]

Duration of mechanical systole. Right and left ventricular mechanical systole were normal in Cases Nos. 1 and 4; both systoles were prolonged in Case No. 5. In Case No. 2 prolonged right ventricular mechanical systole was found when WPW was absent but not when it was present. In Case No. 3 mechanical systole was normal in the left ventricle but prolonged in the right ventricle. Activation of the right ventricle oc-

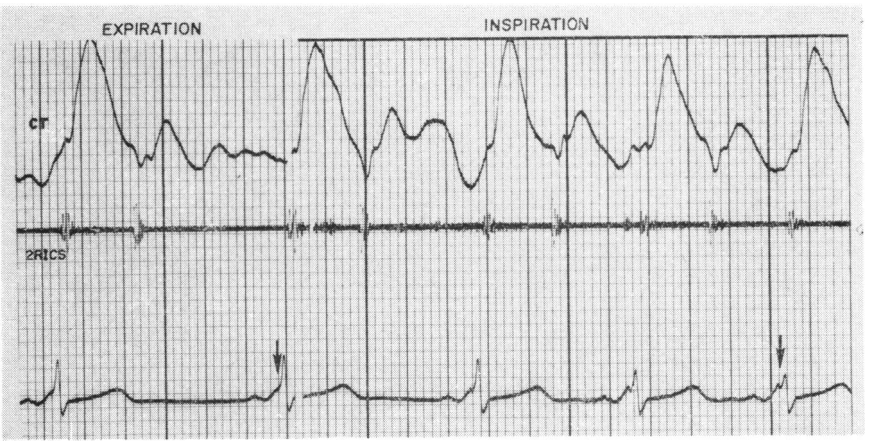

Fig. 4. Tracing obtained in Case No. 3 demonstrating the increase in amplitude and duration of the delta wave in the electrocardiogram on inspiration with diminution of the R wave. Opposite effects are seen on expiration.

curred earlier than normal in all except Case No. 3 (Table III).

The delta wave increased in amplitude and duration during inspiration and decreased on expiration in all cases (Figure 4) except No. 1, where the opposite occurred.

Discussion

The existence of two types of WPW suggests two different courses for spread of excitation and location of the preexcitation area, as postulated by Rosenbaum.[14] Sodi-Pallares[15, 16] considers that the abnormal focus of excitation is located in the right side of the septum, higher for type A and lower for type B; while Hecht[17] and Gamboa[18] believe that in type A the preexcitated area is localized in the left side of the interventricular septum and, in type B, in the right side of the interventricular septum.

Mechanical events in the ventricles might be changed in the presence of WPW and might be detected by analysis of the splitting of the heart sounds. Upon analyzing the sequence of ventricular contraction in the five cases in this study, it was found that contraction of the right ventricle occurred earlier than normal in all cases except Case No. 3. This finding favors the concept that the location of the preexcitation area was in the right ventricle. The absence of ventricular asynchronism in Case No. 3, despite earlier activation of the right ventricle, may be explained by postulating that the preexcitation area was distant from

the conductive system. Although earlier depolarization of this area occurred, ventricular excitation proceeded in both ventricles through the normal pathway.

The earlier excitation and contraction of the right ventricle in these cases, imposed by the WPW syndrome, with the consequent changes in splitting of the heart sounds, would have made it difficult if not impossible to use splitting of the heart sounds for the diagnosis of the associated type of heart disease.

In Case No. 1 there was paradoxical splitting of the heart sounds, as also described recently by Zuberbuhler and Baversfeld[5] in normal children with type B WPW syndrome.

In Case No. 2, despite the fact that the patient had mild infundibular pulmonic stenosis, as proved by cardiac catheterization, when WPW was present the second sound appeared as a single sound. The single sound appears to have been due to earlier activation of the right ventricle rather than to absence of the pulmonic component of the second sound, inasmuch as five years later, in the absence of WPW, physiological splitting of the second heart sound was present (Figure 3).

In Case No. 3, for the reasons previously discussed, no ventricular asynchronism was created by the WPW. The patient had mild aortic insufficiency, which did not produce significant hemodynamic changes, and physiological splitting of the heart sounds was present.

In Case No. 4, with severe mitral insufficiency, wide splitting of the second heart sound would have been expected, as demonstrated by Perloff and Harvey.[19] This patient, however, had a single second heart sound. Similar findings were present in Case No. 5, despite the presence of moderate congenital mitral insufficiency.

In view of the lack of correlation between the splitting of the heart sounds and the type of splitting expected from the anatomical condition present, it was decided to correlate the splitting of the heart sounds with different factors responsible for the creation of ventricular asynchronism in WPW.

The orientation of the delta vector in the frontal plane determines the type of ventricular conduction defect and therefore the type of ventricular asynchronism.[20] When the splitting of the heart sounds was correlated with the orientation of the delta vector in the frontal plane, it was found that Case No. 1, in which the delta vector had the maximum superior orientation, also presented the maximum degree of ven-

tricular asynchronism. However, no difference in ventricular asynchronism was found in Cases Nos. 2, 4, and 5, despite the different orientation of the delta vector in this plane (Table II).

When the same correlation was made with the orientation of the delta vector in the horizontal plane, it was found that in all patients in whom posterior orientation was present ventricular asynchronism was found (Cases Nos. 1, 2, 4, and 5).

Case No. 3, in which the delta vector was oriented anteriorly, presented normal splitting of the heart sound. Although, as postulated by Grant and his co-workers,[20] the orientation of the delta vector in the frontal plane seems to be significant in the production of ventricular asynchronism, the orientation in the horizontal plane also plays a significant role.

The variability in the duration of the P-R interval and the prolongation or shortening of the QRS complex (concertina effect) have been well known since the first description by Ohnell.[21] Transmission of the depolarization wave through the A-V node and the abnormal pathway seems to be influenced by the vagus nerve.[22] Since the vagus effect upon the heart can be studied during normal respiration,[23] it was decided to analyze the changes in amplitude and duration of the delta wave during respiration.

The delta wave increased in duration and amplitude on inspiration in all cases except Case No. 1, in which the opposite changes occurred. This difference of the delta wave during respiration seems to be determined by variation in the effect of the vagus nerve on the abnormal pathway. Fox[24] demonstrated that the delta wave is under the control of the vagus nerve; the administration of atropine sulphate produced significant decrease in the amplitude of this wave. He considered that functional dependence between height of the delta wave and the width of the QRS complex should exist. Lamb[25] has demonstrated that conduction through the abnormal pathway can be changed in certain individuals by breath holding, probably through vagal influences.

In the present study it was found that in the four cases in which the increase in amplitude and duration of the delta wave occurred on inspiration, normal splitting of the heart sounds (Case No. 3) or single second heart sound (Cases Nos. 2, 4, 5) was present, while in Case No. 1, in which opposite changes occurred, greater ventricular asynchronism was present; this led to paradoxical splitting of the heart sounds.

Accordingly it appears that if the delta wave increases in duration and amplitude on expiration, greater ventricular asynchronism can be expected. A tentative explanation is offered. If the vagus nerve has the same effect on the A-V node and on the abnormal pathway, despite the increase in amplitude and duration of the delta wave with inspiration, the faster transmission through the A-V node would preclude greater ventricular asynchronism. However, if the vagus nerve has less effect on the abnormal pathway than on the A-V node, there would be more depolarization through the aberrant pathway with expiration, leading to greater ventricular asynchronism.

The data presented demonstrate that splitting of the heart sounds in WPW depends upon various factors not necessarily related to the anatomical condition of the heart. This imposes serious limitations on the value of splitting of the heart sounds in the diagnosis of heart disease when WPW is present.

Summary

Five children with type B Wolff-Parkinson-White syndrome were studied. One had a normal heart. Two had congenital heart disease (infundibular pulmonic stenosis, mitral insufficiency). Two had rheumatic heart disease (aortic insufficiency, mitral insufficiency).

Splitting of the heart sounds was correlated with: 1) the expected splitting of the heart sounds produced by the anatomical situation present; 2) orientation of the delta vector in the frontal and horizontal plane; 3) changes in amplitude and duration of the delta wave with respiration.

It was demonstrated that each of these factors could produce changes in the sequence of the mechanical contraction of the ventricles, with consequent changes in the splitting of the heart sounds.

It is concluded that changes in the splitting of the heart sounds produced by the Wolff-Parkinson-White syndrome can lead to erroneous diagnosis of heart disease in otherwise normal hearts as well as to mistaken diagnosis in the presence of heart disease.

REFERENCES

1. Wolff, L., Parkinson, J. and White, P. D. Bundle branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia. *Amer. Heart J. 5*:685, 1930.
2. Engle, M. A. Wolff-Parkinson-White syndrome in infants and children. *Amer. J. Dis. Child. 84*:692, 1952.
3. Schiebler, G. L., Adams, P. and Anderson, R. C. The Wolff-Parkinson-White

syndrome in infants and children. A review and a report of 28 cases. *Pediatrics 24*:585, 1959.

4. Swiderski, J., Lees, M. H. and Nadas, A. S. Wolff-Parkinson-White syndrome in infancy and childhood. *Brit. Heart J. 24*:561, 1962.
5. Zuberbuhler, J. R. and Baversfeld, S. R. Paradoxical splitting of the second heart sound in the Wolff-Parkinson-White syndrome. *Amer. Heart J. 70:* 595, 1965.
6. Massie, E. and Walsh, T. J. Ventricular pre-excitation (Wolff-Parkinson-White syndrome.) In: *Clinical Vectorcardiography and Electrocardiography,* 1st ed., vol. 1. Chicago, Year Book, 1960, p. 480.
7. Bleifer, S., Khan, M., Grishman, A. and Donoso, E. Wolff-Parkinson-White syndrome. A vectorcardiographic, electrocardiographic and clinical study. *Amer. J. Cardiol. 4*:321, 1959.
8. Frank, E. An accurate, clinically practical system for spatial vectorcardiography. *Circulation 13*:737, 1956.
9. March, H. W., Selzer, A. and Hultgren, H. N. The mechanical consequences of anomalous atrioventricular excitation (WPW syndrome). *Circulation 23*:582, 1961.
10. Castle, R. R. and Jones, K. L. The mechanism of respiratory variations in splitting of the second heart sound. *Circulation 24*:180, 1961.
11. Gray, I. R. Paradoxical splitting of the second heart sound. *Brit. Heart J. 18:* 21, 1956.
12. Aravanis, C., Lekos, D., Vorides, E. and Michaelides, G. Wolff-Parkinson-White syndrome. Right ventricular precontracting area proved by cardiac catheterization. *Amer. J. Cardiol. 13:* 77, 1964.
13. Bergland, J. L., Rucker, W. R., Reeves, J. T. and Surawicz, B. Pre-excitation as a cause of appearance and increased intensity of systolic murmurs. *Circulation 33*:131, 1966.
14. Rosenbaum, F. F., Hecht, H. H., Wilson, F. N. and Johnson, F. D. The potential variations of the thorax and the esophagus in anomalous atrioventricular excitation (Wolff-Parkinson-White syndrome). *Amer. Heart J. 29*:281, 1945.
15. Sodi-Pallares, D., Soberon, J., Thomsen, P., Fishleder, B. L. and Estandia, A. Contribución al estudio del sindrome de WPW por las derivaciónes intracavitarias. *Arch. Inst. Cardiol. Mex. 18*:1, 1948.
16. Sodi-Pallares, D., Isteni, A. and Medrano, G. Estudios sobre el sindrome de Wolff-Parkinson-White. La activación en el sindrome Wolff-Parkinson-White. *Arch. Inst. Cardiol. Mex. 25*:676, 1955.
17. Hecht, H. H. Further observations on the spread of ventricular excitation. Panel discussion on anomalous atrioventricular excitation. *Ann. N.Y. Acad. Sci. 65*:841, 1957.
18. Gamboa, R., Penaloza, D., Sime, F. and Banchero, N. The role of the right and left ventricle in the ventricular preexcitation (WPW) syndrome. *Amer. J. Cardiol. 10*:650, 1962.
19. Perloff, J. K. and Harvey, W. P. Auscultatory and phonocardiographic manifestations of pure mitral regurgitation. *Progr. Cardiov. Dis. 5*:172, 1962.
20. Grant, R. P., Tomlison, F. B. and Van Buren, J. K. Ventricular activation in the pre-excitation syndrome (Wolff-Parkinson-White). *Circulation 18*:355, 1958.
21. Öhnell, R. F. Pre-excitation, a cardiac abnormality. *Acta Med. Scand. Suppl. 152*:1, 1944.
22. Barker, J. M. Anomalous atrioventricular conduction. In: *The Unipolar Electrocardiogram,* vol. 1. New York, Appleton-Century-Crofts, 1952, p. 548.
23. Katz, L. N. and Pick, A. Disturbances of rhythm in the pre-excitation (WPW) syndrome. In: *Clinical Electrocardiography. I. The Arrhythmias,* vol. 1. Philadelphia, Lea, Febiger, 1958, 679 pp.
24. Fox, T. On the morphology of the Delta wave in the Wolff-Parkinson-White syndrome. *Cardiologia 42*:377, 1963.
25. Lamb, L. E. Respiratory maneuvers in converting WPW syndrome to normal conduction and converting normal conduction to WPW syndrome. *Amer. Heart J. 55*:174, 1958.